

## LETTERS AND CORRESPONDENCE

Letters and correspondence submitted for possible publication must be identified as such. Text length must not exceed 500 words and five bibliographic references. A single concise figure or table may be included if it is essential to support the communication. Letters not typed double-spaced will not be considered for publication. Letters not meeting these specifications will not be returned to authors. Letters to the Editor are utilized to communicate a single novel observation or finding. Correspondence is to be used to supplement or constructively comment on the contents of a publication in the journal and cannot exceed the restrictions for Letters to the Editor. The Editor reserves the right to shorten text, delete objectionable comments, and make other changes to comply with the style of the journal. Permission for publication must be appended as a postscript. Submissions must be sent to Marcel E. Conrad, M.D., Associate Editor, American Journal of Hematology, USA Cancer Center, Mobile, Alabama 36688 to permit rapid consideration for publication.

### Antiplatelet Antibodies in Childhood Idiopathic Thrombocytopenic Purpura

*To the Editor:* I have read the paper by Taub et al. entitled "Characterization of Autoantibodies Against the Platelet Glycoprotein Antigens IIb/IIIa in Childhood Idiopathic Thrombocytopenic Purpura" in the February issue of the *Journal*.

The authors fail to examine the largest study about platelet antibodies (APAs) in childhood acute and chronic idiopathic thrombocytopenic purpura (ITP), published in this *Journal* by ourselves [1]. APAs were shown in the sera of all 103 patients with acute ITP and 42 patients with chronic ITP during thrombocytopenic phase. APA levels were decreased in all patients during recovery, in acute as well as chronic cases, but disappeared completely in none of them. These findings were confirmatory of our earlier results, in which platelet survival was shown to be reverse-correlated with antibody levels in remission [2]. This last point was emphasized, because the authors are planning to study this relation. We have also indicated elevation of APA levels with relapses, though our methodology was different [1]. Because of the presence of APAs in all acute and chronic ITP cases, we strongly suggested that the pathogenesis of acute and chronic ITP were not different [1,2].

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#### REFERENCE

1. Özsoylu Ş, Karabent A, Irken G, Tuncer M: Antiplatelet antibodies in childhood idiopathic thrombocytopenic purpura. *Am J Hematol* 36:82–85, 1991.
2. Özsoylu Ş, Allahverdi H, Laleli Y, Pinar A: Platelet survival in childhood idiopathic thrombocytopenic purpura in remission. *J Pediatr* 89:388–390, 1976.

### Authors' Reply

*To the Editor:* The current trend in examining autoantibodies in idiopathic thrombocytopenia purpura (ITP) involves the use of antigen capture techniques, using either the MAIPA or immunobead assay [1]. Since the initial report by van Leeuwen in 1982 that the glycoprotein IIb/IIIa complex was the predominant platelet antigen at which autoantibodies in chronic ITP were directed [2], numerous studies have examined the antigen specificity of anti-platelet antibodies [3].

The purpose of our study was to determine whether the detection of serum antibodies directed against the platelet glycoprotein complex IIb/IIIa, utilizing the indirect MAIPA assay, differed between cases of acute and chronic ITP [3]. We found no significant difference in antibody detection between the two groups. In our discussion of the study's findings, we focused our comparisons on other studies utilizing current techniques for antibody detection, and, in particular, on studies which examined anti-GP IIb/IIIa antibodies.

We acknowledge Dr. Özsoylu's fine work on childhood ITP, though we did not reference his studies for the above reasons.

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#### REFERENCES

1. Mueller-Eckhardt C, Kiefel V, Santoso S: Recent trends in platelet antigen/antibody detection. *Blut* 59:35, 1989.
2. van Leeuwen CT, van der Ven JTM, Engelhart OP, van den Boire ACGK: Specificity of autoantibodies in autoimmune thrombocytopenia. *Blood* 59:23, 1982.
3. Taub JW, Warriar I, Holtkamp C, Beardsley DS, Lusher JM: Characterization of autoantibodies against the platelet glycoprotein antigens IIb/IIIa in childhood idiopathic thrombocytopenia purpura. *Am J Hematol* 48:104, 1995.

### Reversible Renal Impairment During Leukocytosis Induced by G-CSF In Non-Hodgkin's Lymphoma

*To the Editor:* Granulocyte colony-stimulating factor (G-CSF) has been widely used for various clinical purposes, including the acceleration of neutrophil recovery following chemotherapy and the mobilization of hematopoietic stem cells. Adverse effects of G-CSF have been reported to be few, except for mild bone pain and a few abnormal laboratory findings, including increased serum levels of LDH and alkaline phosphatase [1,2]. We report on a case with non-Hodgkin's lymphoma showing elevated serum creatinine levels during G-CSF therapy.